51. The human TNFα molecule according to claim 50, wherein said TNFα molecule is substantially free from TNFα activity.

52. The human TNFα molecule according to claim 50, wherein the substitution has been made in regions of the unmodified TNFα molecule so as to essentially preserve the β-sheet structure of the B and G strands.

53. The human TNFα molecule according to claim 50, wherein the substitution has been made in regions of the unmodified TNFα molecule which involves the strands of the front β-sheets or the connecting loops so as to essentially preserve the β-sheet structure of any of the strands of the back β-sheet.

54. The human TNFα molecule according to claim 50, wherein the substitution has been made in regions of the unmodified TNFα molecule which involve a segment of the D strand of the back β-sheet.

55. The human TNFα molecule according to claim 50, wherein the substitution comprises at least a segment of the H strand of the front β-sheet and of the connecting loop to the I strand.

56. The human TNFα molecule according to claim 55, wherein the substitution comprises amino acids 132 to 146.

57. The human TNFα molecule according to claim 50, wherein the substitution comprises segments of the H and I strands and the entire connecting loop.

58. The human TNFα molecule according to claim 50, wherein the substitution comprises a segment of the D strand, at least a segment of the E strand, and the entire connecting loop.

59. The human TNFα molecule according to claim 58, wherein the substitution comprises amino acids 65 to 79 or 64 to 84.

60. The human TNFα molecule according to claim 50, wherein the substitution comprises the entire C' and C strands and a segment of the D strand.

61. The human TNFα molecule according to claim 60, wherein the substitution comprises amino acids 40 to 60.

62. The human TNFα molecule according to claim 50, wherein the substitution comprises at least a segment of the E strand and of the front β-sheet of one or both of the connecting loops.

63. The human TNFα molecule according to claim 62, wherein the substitution comprises amino acids 76 to 90.

64. The TNFα according to claim 50, having the amino acid sequence shown in SEQ ID NO:8.

65. The TNFα according to claim 50, having the amino acid sequence shown in SEQ ID NO:10.

66. The TNFα molecule according to claim 50, having the amino acid sequence shown in SEQ ID NO:4 or SEQ ID NO:16.

67. The TNFα according to claim 50, having the amino acid sequence shown in SEQ ID NO:20.

68. The TNFα according to claim 50, having the amino acid sequence shown in SEQ ID NO:14.

69. Dimers, oligomers, or multimers of the human TNFα molecule according to claim 50.

70. The human TNFα molecule according to claim 50 in the fonn of a fusion protein with an adjuvant molecule.

71. The human TNFα molecule according to claim 70, wherein the adjuvant molecule is an immunologically active adjuvant.

72. The human TNFα molecule according to claim 70, wherein the adjuvant molecule is GM-SCF, HSP70 or interleukin.

73. A vaccine against TNFα , comprising an immunogenic amount of one or more human TNFα molecules according to claim 50 in combination with a pharmaceutically acceptable excipient and optionally a pharmaceutically acceptable adjuvant.

74. The vaccine according to claim 73, wherein the excipient is for oral or parenteral administration.

75. The vaccine according to claim 74, wherein the excipient is for subcutaneous, intramuscular, or intradermal administration.

76. The vaccine according to claim 73, wherein the pharmaceutically acceptable adjuvant is aluminum phosphate, aluminum hydroxide, calcium phosphate, muramyl dipeptide, or iscom.--

REMARKS

New claims 50-76 are presented. Claims 50-76 contain the subject matter of canceled claims 1, 2, 4-16, 19, 26, 27, 32, 40-42, and 47, revised to address the §112, ¶2, rejections of record.

Applicants wish to thank Examiner David Romeo for the courteous consideration rendered to applicants' representative during an interview at the USPTO on 31 August 2000 and during telephone discussions on 3 and 4 October 2000.